Is there a link between Alzheimer's and Lyme disease?

Alzheimer's disease is the most common form of dementia. In Alzheimer's nerve cells in the brain break down. Scientists have suspected that this is caused by the accumulation of certain proteins in the cells. These protein accumulations are called plaques and it is proposed that these destroy the cells and disrupt their communication. As a result, the brain can no longer function properly and the brain cells die.

Share50
Tweet
Pin3
53 Shares

History

Alois Alzheimer was a German neuropathologist and psychiatrist after whom Alzheimer's is named. He was the first doctor to describe the disease process and presented his findings to colleagues at a congress for psychiatrists in Tubingen.

His first description was that of a 50-year-old woman whom he had followed after she was aggravated due to paranoia, increasing sleep and memory problems, aggression and confusion. She eventually died from the effects of the disease 5 years later.

Failing hypotheses

Alzheimer's can have a genetic basis and run in the family. This form arises early in life but there is also a form of Alzheimer's that occurs much more often and makes its appearance late in life.

It is important to notice that the symptoms observed in both forms are almost identical. This indicates that other factors must play a role in explaining the neuro-degenerative disorder than the current hypothesis that only seems to relate to the cases of Alzheimer's where a genetic basis exists.

Between 2002 and 2012, 413 trials failed that researched new drugs and treatments for Alzheimer's disease. These drugs did not have any significant effects on the disease process. This was part of the reason for the pharmaceutical company Pfizer to end its research efforts into new medicines for Alzheimer's disease. The failing drug development seems to indicate that the current hypotheses are at least partially incorrect.

The association with infection

The possibility has emerged that different pathogens can cause or contribute to Alzheimer's disease. Multiple pathogens have been found in the brains of Alzheimer's patients including Herpes simplex virus 1, Chlamydia pneumonia and various spirochetes, including Borrelia spirochetes that cause Lyme disease.

Alois Alzheimer and his colleagues already discussed the possibility that pathogens played a role in the formation of the protein accumulations they saw in the brains of these patients 100 years ago. Just like some of the topics surrounding Lyme disease, an infectious component of Alzheimer's remains highly controversial, despite the studies that lead into this direction.

Beta amyloid

Beta amyloid and tau proteins are the specific proteins that play a role in the development of plaques and tangles that are found in the brains of Alzheimer's patients. These proteins have been used as biological markers. Whether these proteins are the cause or a consequence of the disease is still not understood.

Some scientists think that these proteins are indicators of an infectious process. Beta amyloid forms the main component of the plaques in the brains of Alzheimer's patients and recent research has shown that beta amyloid has a potent antimicrobial effect against bacteria, viruses and fungi. Another protein, beta defensin-1, which is part of the innate immune system, also plays a role in the early phase of Alzheimer's disease.

Microglia cells

Microglia cells are the macrophages of the central nervous system. They form the immune system in the brain. They continuously scan for possible damage and foreign substances and remove dead tissue.

Today, microglia cells are considered to be crucial players in innate immune and inflammatory response in multiple neurological disorders, including Parkinson's disease, Multiple Sclerosis, Amyotrophic Lateral Sclerosis and Alzheimer's disease. These microglia cells play an important role in the various inflammatory reactions that are observed in Alzheimer's and that are characteristic of infection.

Syphilitic dementia

The characteristics of syphilitic dementia are very similar to the pathological characteristics of Alzheimer's disease. By means of light microscopy colonies of spirochetes cannot be distinguished from the plaques that play a role in Alzheimer's. In syphilitic dementia brain amyloidosis can also occur, which is similar to the role that beta amyloid plays in Alzheimer's disease.

In syphilitic dementia, Treponema pallidum survives long-term and stationary in the brain and causes chronic infection and inflammation resulting in a slow progressive dementia. Alzheimer's usually develops in later life. This suggests that a slowly growing unconventional infectious organism may be involved in the development. Spirochetes are such unconventional infectious organisms and a series of studies showed that different types of spirochetes can cause dementia in the same way as T. pallidum after a long latent stage.

Oral spirochetes & Borrelia

It is remarkable that the human oral cavity accommodates more than 60 different Treponema species. These were previously considered to be commensal but several of them appear to be predominant and invasive pathogens of which T. pectinovorum, T. amylovorum, T. lecithinolyticum, T. maltophilum, T. medium and T. socranskii were all found in the brains of Alzheimer's patients.

Another spirochete, Borrelia burgdorferi, the causative agent of Lyme disease, is also found in a small percentage of the brains from Alzheimer's patients.

Biofilms

Spirochetes are known to form biofilms and scientists at Drexel University have shown that these biofilms also originate in the brains of patients with Alzheimer's. The mechanism that triggers the formation of biofilms is called quorum sensing. The bacteria sense when they are present in sufficient numbers and then develop a biofilm in response. This biofilm makes them impenetrable for antibiotics and the immune system.

APOE gene

Genes are one of the risk factors for Alzheimer's. One group of Alzheimer's patients have a strong family history of the disease but only 1% directly inherit a gene mutation that causes early-onset Alzheimer's, also known as familial Alzheimer's disease. The gene called APOE can influence your risk for the more common late-onset type of Alzheimer's.

This APOE gene exists in three types: APOE2, E3 and E4. Everyone has two copies of this gene and the combination determines your risk of developing Alzheimer's. E2 is the rarest and carrying a copy of this type reduces your risk of developing Alzheimer's. The E4 allele increases the risk of developing Alzheimer's.

What is more interesting is that possession of the E4 allele has been associated with Multiple Sclerosis as well as with late-onset Alzheimer's. The means by which the APOE allele product might modulate the course of disease in Multiple Sclerosis remains unclear but current opinion holds that it affects the rate of progression to disability rather than the risk for disease development.

Already back in 1911 Multiple Sclerosis was associated with spirochetal infection. When you read the scientific literature between 1911 and 1939 on Multiple Sclerosis you will find that independent scientists from France, Germany and the United Kingdom observed similar things and came to similar conclusions:

- Spirochetes are found in the lesions of the brains of patients who died of Multiple Sclerosis
- These spirochetes can be isolated and can be used to inoculate many animals resulting in the onset of a similar disease

In multiple publications it has been proposed that Chlamydia pneumonia is associated with Multiple Sclerosis. Recent results also indicate that the E4 allele may be involved in neuropathogenesis via its interaction with this bacterial pathogen Chlamydia pneumonia. Thus, the interaction with bacterial pathogens of this E4 allele could also fit in the proposed infectious etiology.

Proposed etiology

Spirochetes reach the brain during a dental procedure or during the dissemination phase of Lyme disease. After a long period of stationary periods alternated with periods of very slow growth, the colony reaches a sufficient population size after which a biofilm is rolled out through quorum sensing. As a result of the biofilm the immune system can no longer reach the infection, which starts to damage the surrounding tissues.

As a result of a cerebrovascular accident the development of Alzheimer's disease is relatively rapid (1-3 years) compared to the time normally required for the onset of the disease (30-50 years). In this situation, the adaptive immune system can penetrate into the brain as a result of the vascular incident where the blood brain barrier function is temporarily compromised. Also the adaptive immune system cannot reach the infection as a result of the biofilm but because the adaptive immune system has a much more powerful response against infection, the damage to surrounding tissues is much greater and the deterioration happens faster.

In this explanation model the presence of the infection, the biofilms or the beta amyloid are not the cause of Alzheimer's. It is rather the reaction of the immune system to these infections that are surrounded by a biofilm, leading to the disease with all its manifestations. Killing the infection before it arrives at the brain or before it can cause damage and create biofilm is extremely important to stop the disease.

Conclusion

We still do not know exactly how Alzheimer's develops or what precisely causes it but an interaction between genetic risk and a number of environmental factors such as bacteria and viral pathogens is suggested by a growing group of scientists. Suspects are Herpes simplex virus 1, Chlamydia pneumonia and different spirochetes such as the oral Treponemes and Borrelia burgdorferi sensu lato.

Applied logic rules out infection as the single cause of Alzheimer's but there must be an interesting interaction between multiple factors. What does this do with our perspective on late stage neurologic Lyme disease?

Share50
Tweet
Pin3
53 Shares

References

Type here to search

Patient Questions

- How accurate are Lyme disease tests?
- Does chronic Lyme disease exist?
- Can Lyme-patients be blood donors?
- Do mosquitoes spread Lyme disease?
- Is there a link between Alzheimer's and Lyme disease?